

Point-of-Care Ultrasound Detection of Acute Myocardial Dysfunction Following Bupropion Overdose: A Case Report

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Abstract

Bupropion overdose is associated with significant morbidity and mortality due to severe neurologic and cardiovascular toxicity, including seizures and life-threatening arrhythmias. Early recognition of electrocardiographic, clinical, and echocardiographic abnormalities is critical for prompt intervention and prevention of clinical deterioration.

We present a case of a 21-year-old female who presented to the emergency department following an intentional polysubstance overdose involving suspected bupropion ingestion. The initial electrocardiogram (EKG) demonstrated sinus tachycardia. However, during continued monitoring, the patient developed QRS widening and seizures. Treatment included benzodiazepines, calcium, and sodium bicarbonate. Point-of-care ultrasound (POCUS) revealed a reduced ejection fraction (EF) of less than 30%, which was later confirmed on formal transthoracic echocardiography. These findings prompted escalation of care, intubation for airway protection and deep sedation, and transfer to the intensive care unit (ICU). During admission, she developed torsades de pointes (TdP), resulting in cardiac arrest. Despite her critical presentation, repeat echocardiography upon resolution of overdose toxicity demonstrated recovery of left ventricular systolic function, with improvement of EF to 50-55% during hospitalization.

This case demonstrates the potential role of POCUS in the early recognition of myocardial dysfunction associated with bupropion toxicity. Together with serial EKG monitoring, these findings identified a patient at high risk for clinical deterioration and supported early escalation of care.

Categories: Substance Use and Addiction, Emergency Medicine

Keywords: bupropion overdose, intentional overdose, point-of-care ultrasound (pocus), torsades de pointes (tdp), wide-qrs

Introduction

Overdose-related presentations are a common cause of emergency department visits and remain a major contributor to morbidity and mortality in the United States [1]. These cases span a wide clinical spectrum, ranging from mild, transient symptoms to life-threatening toxicity requiring advanced interventions such as mechanical ventilation, vasopressor support, and extracorporeal life support.

Bupropion is a norepinephrine-dopamine reuptake inhibitor (NDRI) commonly prescribed for major depressive disorder and smoking cessation. Although bupropion is widely prescribed and generally safe at therapeutic doses, large doses can produce significant neurologic and cardiovascular toxicity [2]. Overdose is characterized by prominent neurologic and cardiovascular effects. Symptom onset may be delayed, particularly with extended-release formulations, often necessitating prolonged observation even in initially stable patients [2,3]. Seizures are a common complication and may occur hours after ingestion, often serving as a key indicator of toxicity severity [3-5].

In addition to neurologic toxicity, bupropion overdose can produce significant cardiotoxic effects, including QRS widening, QT prolongation, conduction abnormalities, and malignant ventricular arrhythmias [4,6]. In severe cases, myocardial depression may lead to cardiogenic shock and cardiac arrest. The proposed mechanisms of toxicity include gap junction blockade and disruption of cardiac ion channel function, not sodium channel blockade, as previously thought [7]. Management is primarily supportive, with benzodiazepines for seizure control and close cardiac monitoring, as seizures can precipitate severe cardiac toxicity due to transient drops in blood pH, allowing more drug to bind to the myocardium [3]. For patients with refractory cardiogenic shock, advanced therapies such as venoarterial extracorporeal membrane oxygenation (ECMO) have been increasingly utilized as a rescue strategy [4,8-10].

Case Presentation

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A 21-year-old female with a past medical history of anxiety, bulimia nervosa, borderline personality disorder, alcohol use disorder, and major depressive disorder presented to the emergency department following an intentional polysubstance overdose. The patient ingested an unknown quantity of bupropion, hydroxyzine, escitalopram, aripiprazole, and ethanol, the most concerning of which was bupropion ingestion. Time from ingestion to ED presentation was approximately 45 minutes. The patient did not have alcohol use disorder; alcohol withdrawal was not a concern.

On initial evaluation, the patient was mildly hypotensive (113/79 mmHg), with notable examination findings including somnolence and slurred speech. Initial EKG demonstrated sinus tachycardia with a QRS of 85 ms and QTc of 415 ms (Figure 1). Laboratory studies were positive for an ethanol level of 226 mg/dL, and UDS was positive for amphetamines and cannabis. Acetaminophen and salicylate levels were undetectable. The initial metabolic panel showed a normal anion gap of 13, magnesium of 1.9 mg/dL, and potassium of 3.4 mmol/L. The patient was initially managed with supportive care. The initial plan for management was to admit the patient for close observation and continuous telemetry monitoring, as she remained persistently tachycardic, increasing her risk for delayed seizures related to bupropion overdose [3].

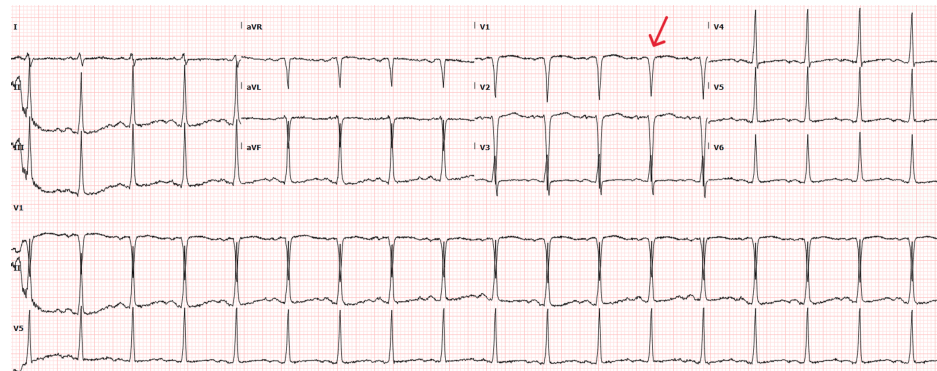


FIGURE 1: Initial EKG showing sinus tachycardia with QRS of 85 ms

During the emergency department course, the patient experienced clinical deterioration characterized by worsening confusion, tachycardia, and abnormal involuntary movements of the extremities, consistent with myoclonus. Aggressive treatment with benzodiazepines was initiated, along with serial EKG monitoring for QRS and QTc changes. Despite treatment, the patient subsequently developed a tonic-clonic seizure that resolved following the administration of 7 mg diazepam. Repeat EKG following seizure activity demonstrated widening of the QRS complex to 153 ms (Figure 2). Two amps of sodium bicarbonate and 2 grams of calcium gluconate were administered, resulting in narrowing of the patient's QRS to 89 ms (Figure 3). POCUS performed shortly thereafter demonstrated severely reduced left ventricular systolic function, with an estimated EF of less than 30% (Video 1). The patient had no previous history of cardiac dysfunction.

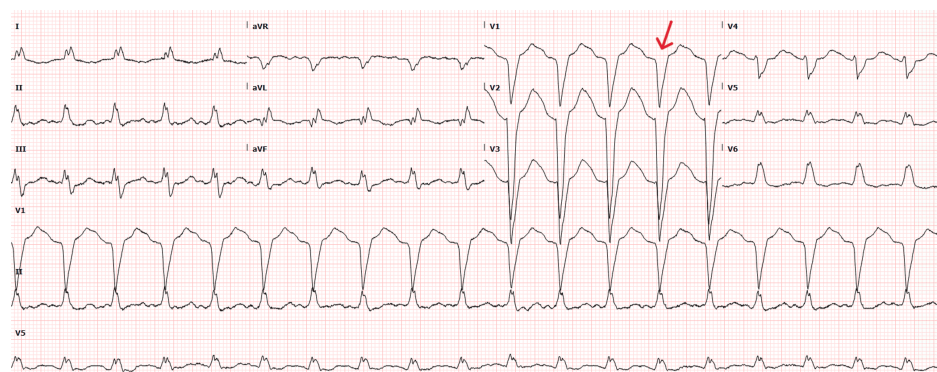


FIGURE 2: Repeat EKG with QRS widening to 153 ms

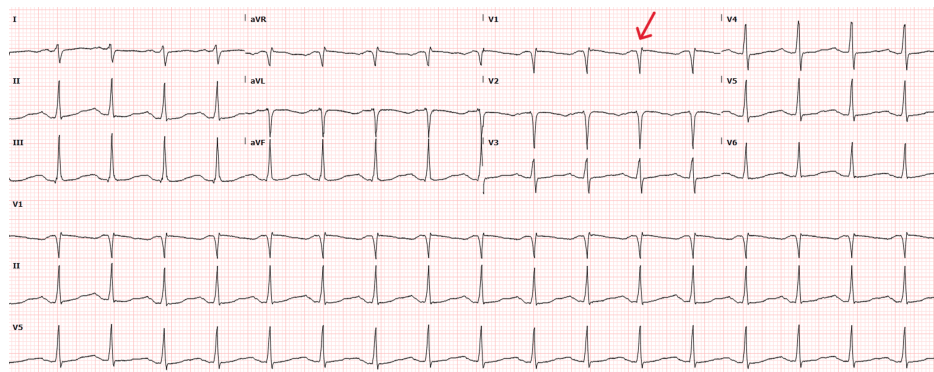


FIGURE 3: Repeat EKG after treatment with improved QRS of 89 ms

VIDEO 1: Point-of-care ultrasound echo video showing reduced left ventricular ejection fraction

View video here: <https://vimeo.com/1212271559?share=copy&fl=sv&fe=ci>

Given the development of significant cardiotoxicity, the patient was subsequently intubated for escalation of deep sedation to prevent further seizures, which were likely contributing to the development of severe cardiac toxicity, and the patient was admitted to the cardiac ICU. Norepinephrine and lipid emulsion therapy were prepared for administration in case the patient developed shock or cardiac arrest.

Following ICU admission, the patient developed polymorphic ventricular tachycardia consistent with TdP and subsequently suffered cardiac arrest. Advanced cardiovascular life support was initiated, and return of spontaneous circulation was achieved after a single 200-J defibrillation, magnesium sulfate administration, and potassium repletion. Formal transthoracic echocardiography demonstrated moderately to severely reduced left ventricular systolic function, with an ejection fraction of 30-35%. Due to concern for progressive cardiogenic shock, the patient underwent ECMO cannulation, and lipids were not administered for fear of interference with the ECMO machine. However, extracorporeal support was ultimately not required.

Over the subsequent 48 hours, cardiac function improved with dobutamine support. Repeat transthoracic echocardiography demonstrated recovery of left ventricular systolic function, with an ejection fraction of 50-55%. The patient was successfully weaned off dobutamine, and repeat echocardiography demonstrated sustained recovery of left ventricular function. The patient was subsequently extubated without complication.

The remainder of the hospital course was notable for continued clinical improvement without recurrent arrhythmias or neurologic deficits. After medical stabilization, the patient was transferred to an inpatient psychiatric unit for further management and was ultimately discharged. Her outpatient antidepressant regimen was changed from escitalopram to sertraline due to the cardiac complications secondary to the recent overdose.

Discussion

Bupropion is commonly used to treat depression, as it is a dopamine agonist and has monoamine reuptake-blocking effects. Bupropion has a phenylethylamine backbone in its chemical structure like that of amphetamines. The resultant concerning adverse effects of overdose are therefore related to its sympathomimetic toxicity, including seizures (CNS) and cardiac toxicity. Other adverse effects include agitation, confusion, tachycardia, hypertension, nausea and vomiting, and hallucinations. Cardiac adverse effects include QRS interval widening, prolonged QTc, and, in severe cases, cardiac arrest, as it inhibits the delayed rectifying potassium channel and gap junction intercellular communication. Myocyte gap junctions are critical to coordinated cardiac conduction and contraction, and blockade of gap junctions by bupropion leads to decreased cardiac output and severe refractory hypotension [7,10].

In this case, the patient took a combination of medications, including unknown amounts of bupropion, hydroxyzine, escitalopram, aripiprazole, and ethanol; her toxidrome, however, was consistent with large-volume bupropion ingestion, possibly potentiated by escitalopram, as her EKG findings and clinical course were consistent with bupropion toxicity. Rapidly obtaining an EKG, and then serial EKGs, in patients with polydrug ingestions is critical to guide management. Also critical in this case was early initiation of benzodiazepines to help mitigate the sympathomimetic effects of bupropion and thereby reduce the risk of

seizures. Notably, agitation, tachycardia, and myoclonus can be warning signs of bupropion overdose as well as predictive of delayed seizures (>8 hours after ingestion). In this patient, these predictive factors helped significantly to guide management. Use of early benzodiazepines, or other sedating medicines like phenobarbital, for persistent tachycardia or agitation is beneficial in attenuating the sympathomimetic aspects of a bupropion overdose, which can precipitate adverse outcomes of bupropion overdose. Also, serial EKGs, especially after a seizure in the setting of bupropion overdose, allowed for rapid detection of the patient's widening QRS, or gap junction blockade, triggering escalation of the patient's care to deep sedation and helping to palliate cardiac toxicity within minutes, as evidenced by the improved EKG.

Also critical in this patient's care path was the use of POCUS. Monitoring the level of reduced ejection fraction in a previously healthy patient who presents to the ED with overdose can rapidly provide beneficial information regarding prognosis and best treatment options. In this case, the fact that POCUS showed an acutely reduced EF in an otherwise healthy patient guided disposition to obtain early ECMO consultation. Without POCUS, this information would not have been readily available, and the patient could have further decompensated without appropriate rescue options.

Additional treatment methods can be very beneficial in bupropion or multiple-substance overdoses to help with management and stabilization. In this patient's case, after the low ejection fraction was noted on POCUS and the decision was made to intubate and transfer the patient to the ICU for ECMO cannulation, stabilization of the patient's hemodynamics was paramount [4,10,11]. Before rapid sequence intubation (RSI) in a patient with a reduced ejection fraction and a widened QRS, it is critical to ensure that the patient's blood pressure can be maintained through the sedation and paralytic medications needed for RSI. In this patient's case, though she needed RSI for airway protection, several steps were taken between the decision to intubate and the procedure itself. Bicarbonate and calcium were given to help mitigate transient acidosis from the patient's seizure, which likely precipitated worsening gap junction blockade with bupropion and cardiac toxicity; a norepinephrine drip was readied, multiple sites of IV access were obtained, and lipid infusions were also prepared. All these steps being performed prior to the use of RSI medications gave this patient the optimal likelihood of remaining hemodynamically stable during RSI despite her cardiac toxicity and reduced ejection fraction. Proceeding with RSI before cardiac and hemodynamic stabilization is considered and optimized can raise the risk of the patient becoming dangerously hypotensive or even experiencing hypotension and cardiac arrest immediately following RSI.

TdP risk management is also critical to keep in mind when managing patients experiencing cardiac toxicity from an overdose of multiple medications. Rapid infusion of magnesium and use of defibrillation allowed this patient's cardiac rhythm to stabilize rapidly despite experiencing a run of pulseless TdP [12]. Furthermore, potassium and magnesium monitoring and replenishment are essential to keep in mind for prolonged QTc risk and, therefore, risk of TdP [12].

Through this combination of methods, the patient was able to be stabilized and treated despite several pathological bupropion overdose complications, including seizures, widened QRS, decreased ejection fraction, and TdP. Once the patient was extubated and stabilized, further care was transferred to the psychiatric unit, where she continued to improve and was ultimately discharged. Her escitalopram prescription was switched to sertraline due to the cardiac complications secondary to the recent overdose, thereby combining and optimizing care of the patient physiologically and psychiatrically to provide her with the best possible opportunity for long-term sustained recovery.

Conclusions

A young female initially arrived mildly tachycardic after an overdose of multiple medications, including bupropion, as a result of a suicide attempt. The patient decompensated and experienced seizures, widened QRS, and decreased EF on POCUS. ECMO was considered given the acute decrease in EF seen on POCUS and wide QRS interval seen on repeat EKG after decompensation. The patient was cannulated for ECMO the following day after a TdP arrest. The patient was closely monitored and provided supportive care in the ICU, following which the patient stabilized. The patient was then successfully extubated and ultimately transferred to the psychiatric unit for further management, after which she was discharged home 18 days after her initial overdose. This case highlights the importance of early detection of warning signs of impending deterioration following overdose, such as EKG and POCUS monitoring, and a wide variety of methods to improve ED management of patients who present following intentional ingestion to allow for optimal care of these complex cases.

Additional Information

Author Contributions

All authors have reviewed the final version to be published and agreed to be accountable for all aspects of the work.

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Disclosures

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